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**UNITED STATES PATENT AND TRADEMARK OFFICE**

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**BEFORE THE BOARD OF PATENT APPEALS  
AND INTERFERENCES**

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*Ex parte* KEVIN Z. QU and ANTHONY SFERRUZZA

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Appeal 2008-3490  
Application 10/714,508  
Technology Center 1600

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Decided: November 12, 2008

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Before ERIC GRIMES, RICHARD M. LEBOVITZ, and MELANIE L.  
McCOLLUM, *Administrative Patent Judges*.

McCOLLUM, *Administrative Patent Judge*.

**DECISION ON APPEAL**

This is an appeal under 35 U.S.C. § 134 involving claims to a genotype determination method. The Examiner has rejected the claims as obvious. We have jurisdiction under 35 U.S.C. § 6(b). We reverse.

**STATEMENT OF THE CASE**

The angiotensin converting enzyme (ACE) “gene is present in the population as different allelic variants. A variant of particular interest

clinically is the presence or absence of a 287 base pair ('bp') non-coding fragment within Intron 16." (Spec. ¶ 0005.) "When this 287 bp sequence is present in an ACE gene, the genotype is designated 'I' for 'insertion'; conversely, when this 287 bp sequence is absent in an ACE gene, the genotype is designated 'D' for 'deletion'" (*id.*). "Because the genome contains two copies of each gene, referred to as 'alleles,' possible ACE genotypes with regard to this variant are D/D, I/D, and I/I" (*id.*).

Claims 2 and 40-72 are pending and on appeal. We will focus on claim 2, which reads as follows:

2. A method of determining an angiotensin converting enzyme (ACE) genotype in a sample, comprising:

amplifying DNA in a single amplification reaction from the sample with a first pair of flanking primers that hybridize to nucleic acid sequences flanking an ACE gene sequence, and a third primer that specifically binds to said ACE gene sequence and together with one of the flanking primers forms a second pair of primers; and

detecting a homozygous ACE genotype by the production of one or two amplification products and a heterozygous ACE genotype by the production of three amplification products.

Claims 2, 40-48, 50, and 67-70 stand rejected under 35 U.S.C. § 103(a) as obvious over Lindpaintner<sup>1</sup> in view of Lin<sup>2</sup> (Ans. 3).

Claims 2, 40-48, 50, and 67-70 stand rejected under 35 U.S.C. § 103(a) as obvious over Teranishi<sup>3</sup> in view of Lin (*id.* at 7).

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<sup>1</sup> Klaus Lindpaintner et al., *A Prospective Evaluation of an Angiotensin-Converting-Enzyme Gene Polymorphism and the Risk of Ischemic Heart Disease*, 332 THE NEW ENGLAND JOURNAL OF MEDICINE 706-711 (1995).

<sup>2</sup> Mei-Hui Lin et al., *Real-time PCR for rapid genotyping of angiotensin-converting enzyme insertion/deletion polymorphism*, 34 CLINICAL BIOCHEMISTRY 661-666 (2001).

Claims 49 and 54-59 stand rejected under 35 U.S.C. § 103(a) as obvious over Lindpaintner in view of Lin, Soubrier,<sup>4</sup> and Buck<sup>5</sup> (*id.* at 11).

Claims 62-65 stand rejected under 35 U.S.C. § 103(a) as obvious over Lindpaintner in view of Lin and van Bockxmeer<sup>6</sup> (*id.* at 15).

Claim 66 stands rejected under 35 U.S.C. § 103(a) as obvious over Lindpaintner in view of Lin, van Bockxmeer, Soubrier, and Buck (*id.* at 18).

Claims 2, 40-48, 50, and 67-70 stand rejected under 35 U.S.C. § 103(a) as obvious over Lin in view of Hiratsuka<sup>7</sup> (*id.* at 21).

Claims 49 and 51-61 stand rejected under 35 U.S.C. § 103(a) as obvious over Lin in view of Hiratsuka, Soubrier, and Buck (*id.* at 25).

Claims 62-65 stand rejected under 35 U.S.C. § 103(a) as obvious over Lin in view of Hiratsuka and van Bockxmeer (*id.* at 33).

Claim 66 stands rejected under 35 U.S.C. § 103(a) as obvious over Lin in view of Hiratsuka, van Bockxmeer, Soubrier, and Buck (*id.* at 36).

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<sup>3</sup> Megumi Teranishi et al., *Insertion/deletion angiotensin converting enzyme gene polymorphism affects the microvascular structure of the kidney in patients with nondiabetic renal disease*, 17 JOURNAL OF HYPERTENSION 351-356 (1999).

<sup>4</sup> US 5,736,323, Apr. 7, 1998.

<sup>5</sup> G.A. Buck et al., *Design Strategies and Performance of Custom DNA Sequencing Primers*, 27 BIOTECHNIQUES 528-536 (1999).

<sup>6</sup> Frank M. van Bockxmeer et al., *Angiotensin-Converting Enzyme and Apolipoprotein E Genotypes and Restenosis After Coronary Angioplasty*, 92 CIRCULATION 2066-71 (1995).

<sup>7</sup> Masahiro Hiratsuka et al., *Detection of Angiotensin-Converting Enzyme Insertion/Deletion Polymorphisms Using Real-Time Polymerase Chain Reaction and Melting Curve Analysis with SYBR Green I on a GeneAmp 5700*, 289 ANALYTICAL BIOCHEMISTRY 300-303 (2001).

Claims 71 and 72 stand rejected under 35 U.S.C. § 103(a) as obvious over Lin in view of Hiratsuka (*id.* at 39).

#### LINDPAINTNER OR TERANISHI WITH LIN

The Examiner rejects claims 2, 40-48, 50, and 67-70 under 35 U.S.C. § 103(a) as obvious in view of Lindpaintner and Lin and as obvious in view of Teranishi and Lin. The Examiner also rejects claims 49, 54-59, and 62-66 under 35 U.S.C. § 103(a) as obvious in view of Lindpaintner and Lin and further in view of one or more of Soubrier, Buck, and van Bockxmeer.

The Examiner relies on both Lindpaintner and Teranishi for teaching:

a method of determining an angiotensin converting enzyme genotype in a sample comprising: a) amplifying DNA from the sample with a i) first pair of flanking primers that hybridize to nucleic acid sequences flanking an ACE gene sequence, and ii) a third primer that specifically binds to said ACE gene sequence and together with one of the flanking primers forms a second pair of primers . . . ; and b) detecting a[n] . . . ACE genotype by the production of . . . amplification products.

(*Id.* at 4 and 7-8.) The Examiner finds that neither Lindpaintner nor Teranishi “teach that the amplification reaction is carried out with three separate primers in a single amplification reaction” (*id.* at 6 and 10).

The Examiner relies on Lin for teaching “amplification using three primers simultaneously in a single amplification reaction” (*id.*). In particular, the Examiner finds that “Lin teaches a method of determining an angiotensin converting enzyme (ACE) genotype in a sample comprising: amplifying DNA in a single amplification reaction from a sample” (*id.*).

The Examiner concludes that it would have been *prima facie* obvious “to have incorporated the genotyping analysis of the ACE genotype, with three primers included in a PCR reaction simultaneously taught by Lin into

the method of ACE genotyping with Lindpaint[n]er [or Teranishi]" (*id.*). In particular, the Examiner finds:

Considering the potential for high throughput analysis using the real-time and multiplexed method taught by Lin and the significant time saving provided by multiplexing the amplification using the three primers simultaneously, it would have been obvious to extend the method of Lindpaint[n]er or Lin to achieve two I amplicons and a single D amplicon in a single amplification reaction.

(*Id.* at 44-45.) In addition, the Examiner finds:

When Teranishi is combined with the three primer, single amplification method taught by Lin, a method that comprises two insertion specific amplicons together with a deletion specific amplicon is achieved. The result of this combination of references yields genotype analysis comprising two amplicons in I allele homozygotes, a single amplicon in D allele homozygotes and three amplicons in I/D heterozygotes.

(*Id.* at 47.)

Appellants contend that none of Lindpaintner, Teranishi, and Lin teaches a method that produces two amplicons from the I-allele in a single PCR reaction (App. Br. 14 and 24). Appellants also contend that there would have been no motivation to combine Lin's real-time PCR method with the two-step methods of Lindpaintner and Teranishi (*id.* at 15 and 24).

#### *Issue*

The issue is whether the Examiner has set forth a *prima facie* case that it would have been obvious to both amplify DNA in a single amplification reaction using three primers and detect a homozygous genotype by the production of one or two amplification products and a heterozygous genotype by the production of three amplification products.

*Findings of Fact*

1. Lindpaintner discloses that the angiotensin converting enzyme (ACE) gene “contains a polymorphism based on the presence (insertion [*I*]) or absence (deletion [*D*]) within an intron of a 287-base-pair (bp) nonsense DNA domain, resulting in three genotypes (*DD* and *II* homozygotes, and *DI* heterozygotes)” (Lindpaintner 706).

2. Lindpaintner discloses determining ACE genotypes by identifying the *D* and *I* alleles based on PCR amplification “of the respective fragments from intron 16 of the *ACE* gene and size fractionation and visualization by electrophoresis” (*id.* at 707).

3. Specifically, Lindpaintner discloses using a “primer pair to amplify the *D* and *I* alleles, resulting in 319-bp and 597-bp amplicons, respectively. . . . The amplification products of the *D* and *I* alleles were identified by 300-nm ultraviolet transillumination as distinct bands.” (*Id.*)

4. In addition, Lindpaintner discloses:

Because the *D* allele in heterozygous samples is preferentially amplified, each sample found to have the *DD* genotype was subjected to a second, independent PCR amplification with a primer pair that recognizes an insertion-specific sequence. . . . The reaction yields a 335-bp amplicon only in the presence of an *I* allele, and no product in samples homozygous for *DD*. . . . This procedure correctly identified the 4 to 5 percent of samples with the *DI* genotype that are misclassified as *DD* with the insertion-spanning primers.

(*Id.* at 707-708.)

5. Teranishi also discloses determining the *I/D*, *II*, and *DD* genotypes of the ACE gene by PCR (Teranishi 352).

6. Teranishi discloses a first PCR reaction using sense and antisense primers, resulting in “a 190 bp fragment in the absence of the insertion and a 490 bp fragment in the presence of the insertion” (*id.*)

7. Teranishi also discloses a second PCR reaction in which “the sense primer was replaced by an insertion-specific primer. . . . A 408 bp PCR product indicates the presence of at least one I allele.” (*Id.*)

8. Lin discloses genotyping the “ACE[ ]gene I/D allele by real-time PCR and melting curve analysis” (Lin 662 (emphasis omitted)).

9. To conduct PCR, Lin discloses adding ACE1, ACE2, and ACE3 primers to the reaction mixture (*id.*).

10. Lin’s ACE1 and ACE3 primers flank an intron 16 sequence of the ACE gene. Lin’s ACE2 primer binds to this ACE intron 16 sequence. (*Id.* at 663: Fig. 1.)

11. Using these primers, Lin discloses the amplification of a 65 bp PCR product for the I allele and an 84 bp PCR product for the D allele (*id.* at 664).

12. Specifically, Lin discloses that “the II alleles produced a distinct melting peak at 73.9°C, while the DD alleles individual generated a melting peak at 76.2°C” (*id.* at 663).

13. With regard to an individual having an I/D allele, Lin discloses forming “two melting peaks at 73.9°C and 76.2°C corresponding to the I and D allele” (*id.* at 662).

14. Based on the size of the I/D insertion (287 bp), Lin’s 65 bp PCR product for the I allele must be the product of the ACE2 and ACE3



primers, not of the ACE1 and ACE3 primers (*id.* at 663: Fig. 1; *see also* Ans. 24 and Reply Br. 9).

15. Lin compares its real-time technique to conventional PCR with “primers situated outside the insertion/deletion area in intron 16 of the ACE gene, [which results in] a PCR product of 490 base pairs (bp) and 190 bp represent[ing] the insertion and deletion allele, respectively” (*id.* at 662).

16. In the context of this conventional PCR, Lin states:

With the smaller size of PCR product, the D allele may be preferentially amplified in the heterozygote individuals giving rise to mistyping of ID individuals in approximately 5% of cases. . . . To avoid this mistyping, each sample found to have the DD genotype was subjected to a second independent PCR amplification with a set of primers that recognize an insertion-specific sequence.

(*Id.*)

17. Lin discloses that, “[w]ith the real-time PCR method, [they] were able to rapidly and accurately complete the genotyping of the 40 specimens in one round of real-time PCR,” including the I/D specimens that had been mistyped as DD by the first amplification step of conventional PCR (*id.* at 663).

18. Lin states that the mistyping “problem arose due to the different size of I and D allele PCR products” (*id.* at 664).

19. However, Lin states that, with their real-time PCR, “the PCR product is relatively small (65 bp for I allele and 84 bp for D allele) and the preferential amplification of either I or D allele is negligible. This potential problem was further overcome by optimization of the primers concentration.” (*Id.*)

*Analysis*

A claim “composed of several elements is not proved obvious merely by demonstrating that each of its elements was, independently, known in the prior art.” *KSR Int’l v. Teleflex Inc.*, 127 S. Ct. 1727, 1741 (2007). The relevant question is “whether there was an apparent reason to combine the known elements in the fashion claimed by the patent at issue.” *Id.*

As admitted by the Examiner (Ans. 6 and 10), neither Lindpaintner nor Teranishi discloses amplifying DNA in a single amplification reaction with a first pair of flanking primers that hybridize to nucleic acid sequences flanking a gene sequence and a third primer that specifically binds to the gene sequence, as recited in claims 2 and 67. Instead, Lindpaintner and Teranishi each discloses a two step amplification, where the first step uses flanking primers to form a D amplicon and a longer I amplicon and the second step uses an internal primer together with another primer to form a shorter I amplicon (Findings of Fact (FF) 2-7). If these two steps were combined in a single amplification reaction under conditions in which all three of these products (and no other products) are formed, the amplification would result in a single amplicon for homozygous DD individuals, two different amplicons for homozygous II individuals, and three amplicons for heterozygous I/D individuals. However, we agree with Appellants that the Examiner has not set forth a prima facie case that it would have been obvious to modify Lindpaintner or Teranishi to combine their two amplification steps into a single amplification reaction under conditions in which all three of these products are formed.

In particular, neither Lindpaintner nor Teranishi teach or suggest combining these two amplification steps into one reaction. Instead, both references disclose that the steps are conducted as separate amplification reactions (FF 4 and 7). In addition, Lindpaintner discloses that the second amplification reaction serves to correctly identify samples with a DI genotype that, due to preferential amplification, are misclassified as DD by the first amplification step (FF 4). The Examiner has not provided an adequate explanation as to whether this purpose can be achieved by combining the amplification steps in a single reaction under conditions in which the heterozygous genotype forms three amplification products.

Lin discloses accurately genotyping specimens in one round of real-time PCR (FF 17). However, Lin discloses detecting one amplification product for each of the II and DD alleles (i.e., the homozygous ACE genotypes) and two amplification products for the I/D allele (i.e., the heterozygous ACE genotype) (FF 11-13). Lin does not disclose the production of three amplicons. As a result, the Examiner has not shown that, if Lindpaintner or Teranishi is modified to include a single amplification reaction using three primers as described in Lin, it would have been obvious to detect a homozygous genotype by the production of one or two amplification products and a heterozygous genotype by the production of three amplification products, as recited in claims 2 and 67. For at least these reasons, we reverse the rejections of claims 2 and 67 and of claims 40-48, 50, and 68-70, which depend from either claim 2 or claim 67, under 35 U.S.C. § 103(a) as obvious in view of Lindpaintner and Lin and as obvious in view of Teranishi and Lin.

Claim 62 is directed to a method for identifying a patient with a heightened risk of suffering from a disease. The method includes determining the ACE genotype in a sample by the steps recited in claim 2. We have already concluded that the Examiner has not set forth a prima facie case that claim 2 would have been obvious in view of Lindpaintner and Lin. The Examiner relies on van Bockxmeer for additional limitations recited in claim 62, and has not pointed to any disclosure in this reference that would make up for the deficiencies discussed above (Ans. 15-17). Thus, we conclude that the Examiner has not set forth a prima facie case that claim 62 and claims 63-65, which depend from claim 62, would have been obvious in view of Lindpaintner, Lin, and van Bockxmeer. We therefore reverse the obviousness rejection of these claims in view of these references.

Claims 49 and 54-59 depend from claim 2 and claim 66 depends from claim 63. We have already concluded that the Examiner has not set forth a prima facie case that claims 2 and 63 would have been obvious over Lindpaintner, Lin, and van Bockxmeer. The Examiner relies on Soubrier and Buck for limitations recited in dependent claims, and has not pointed to any disclosure in these references that would make up for the deficiencies discussed above (Ans. 11-15 and 18-20). Thus, we conclude that the Examiner has not set forth a prima facie case that claims 49, 54-59, and 66 would have been obvious over Lindpaintner and Lin in view of the additional references applied therewith. We therefore reverse the obviousness rejection of these claims in view of these references.

LIN AND HIRATSUKA

The Examiner rejects claims 2, 40-48, 50, and 67-72 under 35 U.S.C. § 103(a) as obvious over Lin in view of Hiratsuka. The Examiner also rejects claims 49 and 51-66 under 35 U.S.C. § 103(a) as obvious over Lin in view of Hiratsuka and further in view of one or more of Soubrier, Buck, and van Bockxmeer.

The Examiner relies on Lin for teaching “amplification using three primers simultaneously in a single amplification reaction for the determination of angiotensin converting enzyme genotype” (Ans. 21). Specifically, the Examiner finds:

Lin teaches a method of determining an angiotensin converting enzyme genotype in a sample comprising: a) amplifying DNA from the sample with a i) first pair of flanking primers that hybridize to nucleic acid sequences flanking an ACE gene sequence, and ii) a third primer that specifically binds to said ACE gene sequence and together with one of the flanking primers forms a second pair of primers . . . ; and b) detecting a homozygous ACE genotype by the production of one or two amplification products.

(*Id.*) The Examiner also finds:

The multiplexed real-time PCR amplification technique taught by Lin genotypes the samples using three primers, however only detects two of the three potential amplification products - the deletion allele, a product of the amplification using ACE1 and ACE3 when the insertion is not present, resulting in an amplicon of 84 base pairs in length and the insertion allele, a product of the ACE2 (insertion specific) primer and ACE3, resulting in an amplicon of 65 base pairs in length.

(*Id.* at 24.)

The Examiner relies on Hiratsuka for teaching “a similar method of genotype analysis and [for] teach[ing] very similar flanking primers which amplify the same general region of the ACE gene sequence” (*id.*). The Examiner finds:

Unlike Lin, Hiratsuka detects two amplicons, both products of the ‘ACE1’ and ‘ACE3’ primers - an insertion allele, where the full length product of the flanking primers produces an amplicon of 373 base pairs in length when the insertion is present, and a deletion allele, where the product of the flanking primer produces an amplicon of 85 base pairs in length when the insertion is not present.

(*Id.*) The Examiner finds that “combining these amplicons with the amplicons provided by Lin would produce three amplification products in the heterozygote - 2 products for the I allele and 1 for the D allele” (*id.* at 23).

The Examiner concludes:

It would have been *prima facie* obvious . . . to have extended the genotyping analysis of the ACE genotype, with three primers included in a PCR reaction simultaneously taught by Lin to incorporate the third amplicon comprising the product of the ACE1 and ACE3 primers in the presence of the 287 bp Alu insertion, resulting in an amplicon of 373 basepairs as taught by Hiratsuka.

(*Id.* at 23-24.) In particular, the Examiner states that it would have been *prima facie* obvious “that the placement of the primers taught by Lin, using the conditions and primer concentrations taught by Hiratsuka would produce a set of amplicons wherein the presence of three amplicons is indicative of a heterozygous genotype with a reasonable expectation for success” (*id.* at 25).

Appellants contend that the “question here is not whether a third amplicon could have been produced by Lin from the ACE1-ACE3 primer pair (clearly it is possible), but rather, whether one would have been motivated to produce such an amplicon and whether it would be useful in Lin’s method” and that “the answer to both questions is no” (Reply Br. 11). In particular, Appellants contend that “there is no motivation to produce a second amplicon from the I-allele because it may confound identification of the characteristic I- and D-allele amplicons already produced, but would not provide any additional information” (*id.*).

*Issue*

The issue is whether the Examiner has set forth a prima facie case that it would have been obvious to modify Lin to detect a heterozygous genotype by the production of three amplification products.

*Findings of Fact*

20. Hiratsuka discloses “the analysis of ACE insertion/deletion polymorphisms” by performing PCR using a forward and reverse primer and melting curve analysis (Hiratsuka 301).

21. Hiratsuka discloses that the resulting “373-bp fragment corresponded to the I allele, whereas the 85-bp fragment corresponded to the D allele” (*id.*).

*Analysis*

As noted above, a claim “composed of several elements is not proved obvious merely by demonstrating that each of its elements was, independently, known in the prior art.” *KSR Int’l v. Teleflex Inc.*, 127 S. Ct. 1727, 1741 (2007). The relevant question is “whether there was an apparent

reason to combine the known elements in the fashion claimed by the patent at issue.” *Id.*

Lin discloses genotyping the “ACE[ ]gene I/D allele by real-time PCR and melting curve analysis” (FF 8). To conduct PCR, Lin discloses adding ACE1, ACE2, and ACE3 primers to the reaction mixture (FF 9). Lin’s ACE1 and ACE3 primers flank an intron 16 sequence of the ACE gene and its ACE2 primer binds to this ACE intron 16 sequence (FF 10). Using these primers, Lin discloses detecting one amplification product for each of the II and DD alleles (i.e., the homozygous ACE genotypes) and two amplification products for the I/D allele (i.e., the heterozygous ACE genotype) (FF 11-13). Lin does not disclose amplifying the I allele with flanking primers ACE1 and ACE3 (FF 14).

Hiratsuka discloses amplifying the I allele with flanking primers (FF 20-21). However, the Examiner has not provided a sufficient reason for one of ordinary skill in the art to vary the primer concentrations and/or other conditions in Lin in order to amplify the I allele with flanking primers ACE1 and ACE3. In particular, Lin specifically discloses optimizing the primer concentrations to overcome preferential amplification between the two PCR products formed in its reaction (FF 19). The Examiner has not explained why one of ordinary skill in the art would have instead used “the conditions and primer concentrations taught by Hiratsuka” (Ans. 25). For at least these reasons, we reverse the rejection of claims 2 and 67 and of claims 40-48, 50, and 68-72, which depend from either claim 2 or claim 67, under 35 U.S.C. § 103(a) as obvious over Lin in view of Hiratsuka.



As discussed above, the method of claim 62 includes determining the ACE genotype in a sample by the steps recited in claim 2. We have already concluded that the Examiner has not set forth a prima facie case that claim 2 would have been obvious over Lin in view of Hiratsuka. The Examiner relies on van Bockxmeer for additional limitations recited in claim 62, and has not pointed to any disclosure in this reference that would make up for the deficiencies discussed above (Ans. 33-36). Thus, we conclude that the Examiner has not set forth a prima facie case that claim 62 and claims 63-65, which depend from claim 62, would have been obvious over Lin in view of Hiratsuka and van Bockxmeer. We therefore reverse the obviousness rejection of these claims in view of these references.

Claims 49 and 51-61 depend from claim 2 and claim 66 depends from claim 63. We have already concluded that the Examiner has not set forth a prima facie case that claims 2 and 63 would have been obvious over Lin, Hiratsuka, and van Bockxmeer. The Examiner relies on Soubrier and Buck for limitations recited in dependent claims, and has not pointed to any disclosure in these references that would make up for the deficiencies discussed above (Ans. 25-32 and 36-39). Thus, we conclude that the Examiner has not set forth a prima facie case that claims 49, 51-61, and 66 would have been obvious over Lin and Hiratsuka in view of the additional references applied therewith. We therefore reverse the obviousness rejection of these claims in view of these references.

CONCLUSION

The Examiner has not shown that the claims would have been obvious to a person of ordinary skill in the art based on the applied references. We therefore reverse the rejections of claims 2 and 40-72.

REVERSED

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